

## OCCASIONAL PAPER

# The 'Ajuda Paralyzes': history of a neuropsychiatric debate in mid-19th-century Portugal

Paulo Fontoura<sup>1,2</sup><sup>1</sup> Department of Immunology, Faculty of Medical Sciences, New University of Lisbon, Portugal<sup>2</sup> Translational Medicine Group CNS, Roche Pharmaceuticals, Basel, Switzerland

Correspondence to: Paulo Fontoura,  
F. Hoffmann – La Roche,  
Pharma Research and Exploratory Development,  
Grenzacherstrasse 124, Building 74 Room 3W.412,  
4070 Basel BS, Switzerland  
E-mail: paulo.fontoura@roche.com; paulo.fontoura@fcm.unl.pt

The second half of the 19th century witnessed an increasing interest in neurology and psychiatry by Portuguese physicians, in parallel with the overall development of these disciplines in other countries. This process is reflected in the numerous case report publications as well as in debates taking place at the Lisbon Society of Medical Sciences, the major scientific forum of that time. The 'Ajuda Paralyzes' were a mysterious succession of epidemics that occurred during 1860–64 in the Ajuda asylum for cholera and yellow fever orphans, which were extensively discussed during 1865–66 by Bernardino Antonio Gomes, Antonio Maria Barbosa, Abel Jordão and Eduardo Motta. Studying this debate helps understand the initial stages of development and the great interest that 'nervous diseases' had for Portuguese clinicians in the mid-19th century and possibly provides one of the first modern descriptions of nutrition-related polyradiculoneuropathy and the ocular findings associated with avitaminosis A. This debate took place at a decisive time for the scientific development of neurology and psychiatry, concurrent with the widespread application of the clinical-anatomical method and neuropathology to the study of diseases of the nervous system, which would set the foundations for our own modern pathophysiological framework. Therefore, the 'Ajuda paralyzes' debate also provides a good basis for a discussion on the evolution of the concepts of hysteria and psychosomatic disease and the description of peripheral neuropathy from among a wealth of other entities that did not withstand the test of science.

**Keywords:** history of neurology; 19th century; Portugal**Abbreviation:** SCML = Sociedade das Ciências Médicas de Lisboa

## Historical context: the great sanitary crisis of 1856–57

During the second half of the 19th century, Portugal underwent a period of sustained growth and political stability after the turmoil of the Civil War (1828–34) and the fractious beginnings of the constitutional monarchy in the period known to historians as the 'Devourism'. From the beginning of the 'Regeneration' period onward (starting in 1851), for the first time a numerous elite of

clinicians and researchers appeared in Portuguese medicine, including several physicians who had been trained and maintained contact with some of the mainstream centres of European academic medicine (especially Paris, where the anatomoclinical method was transforming medical science). The Lisbon Society of Medical Sciences (*Sociedade das Ciências Médicas de Lisboa*, SCML), founded in 1822 and today one of the oldest medical societies in continuing existence, was the privileged forum for scientific discussion during this period and a transforming force for social change. By the mid-19th century, the SCML had acquired

considerable national prestige and a dynamic membership, and its list of presidents includes most of the major figures in Portuguese medicine. The SCML Journal, published from 1836 until the present day, became the main scientific medical publication at that time, where the most relevant original works appeared (Torres Pereira *et al.*, 2006).

Historically, the chief identifiable milestones for the establishment of neurology and psychiatry as independent medical disciplines in Portugal came only with the creation of the Lisbon and Porto Medical Schools in 1911 and their first professorships in these disciplines, given to Nobel prize winner Egas Moniz (1874–1955) and to Magalhães Lemos (1855–1931), respectively (Fontoura, 2008). Previously, however, there were already some signs of activity in these areas. The first dedicated internment unit for mental disorders in the country, the Rilhafoles asylum in Lisbon (now called hospital Miguel Bombarda), had been created in 1848 to replace the markedly unsound conditions in which ‘alienated’ patients had been treated at St. Joseph’s Hospital up until then (Senna, 1885; Ferreira de Mira, 1947). A similar institution was founded in Porto in 1883, the *Conde de Ferreira* hospital for alienated patients (Pereira *et al.*, 2005). During the next six decades, several physicians showed an evident interest in the diseases of the nervous system. Among the better known is Antonio Maria Bettencourt Rodrigues (1854–1933), who trained in Paris with Jean-Martin Charcot (1825–93) and Charles-Joseph Bouchard (1837–1915), gave the first public series of lectures on ‘neuropathology and psychiatry’ in Lisbon in 1887–89 and founded the first journal dedicated to this area, the *Revista de Neurologia e Psychiatria* (Review of Neurology and Psychiatry), which lasted from 1888 to 1890. Other relevant figures include Antonio Maria de Senna (1845–90), the great reformer of psychiatric legislation; Miguel Bombarda (1851–1910) the would-be first president of the Portuguese republic, responsible for the clinic for neurological diseases at St. Joseph hospital (*Clinica externa de doenças nervosas e mentais*), director and reformer of the Rilhafoles asylum from 1892 until his death; and Virgílio Machado (1859–1927), trained at the Salpêtrière with Edouard Brissaud (1852–1909) and Fulgence Raymond (1844–1910), author of the first Portuguese textbook on neurological semiology (*Elementos de Neurosemiologia Clinica*, 1919) and founder of the first neurological diseases ward in St. Joseph’s hospital in Lisbon at the end of the 19th century (Ferreira de Mira, 1947; Reis de Oliveira, 2006; Araujo, 2007; Fontoura, 2008).

The intention of this article is to outline what was undoubtedly one of the great debates on neuropsychiatric issues in 19th-century Portugal, fully absorbing the SCML in 1865–66, and to comment on the observations of this first generation of what could be called Portuguese proto-neurologists. Also, I will endeavour to provide a critical appraisal of the debate in the light of contemporary scientific knowledge, illustrating the depth and sophistication of the discussion as well as proposing a new interpretation for the puzzling clinical phenomena which were described.

The historical setting for this debate was the great sanitary crisis that occurred in Lisbon during 1856–57. In those 2 years, epidemics of cholera and yellow fever ravaged the capital city of Portugal

and together caused up to 10 000 deaths (~2.5% of the population). Lisbon was a prosperous and vibrant city in the mid-19th century and one of the major European ports. However, sanitary conditions in the city were deficient, leading to recurrent public health problems. Yellow fever was not endemic but was regularly imported in cargo ships coming from Brazil; in fact, in 1723 Lisbon suffered the first documented epidemic of this disease in Europe (Eager, 1902; Augustin, 1909b) in which ~6000 people died. In 1856, there was a further outbreak of yellow fever in Lisbon, affecting 122 victims, together with a cholera epidemic [part of the third cholera pandemic (1852–60)], which killed 3275 people (Reino, 1859; Augustin, 1909b). During the following year, the city was again severely hit by this scourge; between 22 July and 10 December, 13 575 cases of yellow fever were registered, with a total of 5652 deaths (mainly young adults between 20 and 40 years old), and it is quite possible that the total number of cases was as high as 18 000 (Reino, 1859; Augustin, 1909b). To put it into context, this is a higher number than for more notorious contemporary epidemics in the southern United States in that period, for example in New Orleans between 1817 and 1899 (Augustin, 1909a; Patterson, 1992). The history of this epidemic was extensively described in a report by the Royal Council of Public Health; according to this source, three ships from Brazil (the *Tamar*, the *Gerona* and the *Cidade de Belem*) were suspected of bearing the disease to Lisbon. In all three vessels there were records of diseased patients, and the index case of the epidemic was a customs worker, presumably in close contact with the ship’s cargo holds in which the mosquito vectors had been transported from Brazil (Reino, 1859).

As could be expected, the epidemics of 1856–57 had a severe impact on the social structure of Lisbon’s population and led to public expressions of panic and religious fervour (Fig. 1). The demise of a significant number of young adults created a whole generation of orphans for whom little social support was available. In response to this emergency, King Pedro V (1837–61) created, in 1856, a new orphan asylum in an abandoned building in the Ajuda quarter under the direction of the St. Vincent de Paul Sisters of Charity and Lazarite priests (Esteves Pereira and Rodrigues, 1904). The Ajuda asylum functioned up until the end of the monarchy in 1910, when such religious institutions were extinguished or reconverted. In its latter days, children were taken in at age 7 and left at 18 years, and endured a ‘[...] paternal and practical education, which prepares the 100 interneers for the harsh chores required of house maids [although some] have studied in the Normal School and obtained a teaching diploma [...]’ (O Instituto, 1904) (Fig. 1). It is not hard to imagine the dramatic circumstances that surrounded the creation and initial years of the Ajuda asylum and the physical and psychological conditions in which the first orphans were admitted. Dozens of young children in shock, disoriented, malnourished, who would have witnessed the gruesome death of one or both parents from such dreadful diseases, were left without means of subsistence and no clear expectations about their future. These orphans were taken into this recently created institution, which still had little to offer besides a roof, some material comfort and a stern religious education, as we will see later.



**Figure 1** (Top) Penitential procession in Lisbon during the 1857 yellow fever epidemic. Reproduction of an original drawing from the second half of the 19th century. (Bottom) Group photograph of an intern class at the Ajuda asylum; illustrated postcard from the late 19th century.

## The 'Ajuda paralyse': the debate and its protagonists

From 1860 until 1864, four mysterious epidemics would strike the orphan population of the Ajuda asylum—referred to as a whole as the 'Ajuda paralyse'. They would be the subject of a 2-year-long debate in the SCML, led by four major participants: Bernardino Antonio Gomes (1806–77), Antonio Maria Barbosa (1825–92), Abel Jordão (1833–74) and Eduardo Augusto Motta (1837–1912) (Fig. 2); all of them would publish at least two works on the subject, one each in 1865 and 1866, roughly splitting into two opposing fields, Gomes and Barbosa versus Jordão and Motta. Bernardino Gomes was the president of the SCML (1864–66 mandate) at the time of the debate and although past his more productive years, was still a renowned figure. Trained in Paris, he had been the first physician to use chloroform and ether anaesthesia in Portugal, and in 1844 had published a landmark report regarding a personal tour of the major establishments for 'alienated' patients in Europe [including institutions in Holland, Germany, France, northern Italy and England (Gomes, 1844)], which provided support for the creation of the Rilhafoles asylum in 1848. He would start the debate, and his contributions

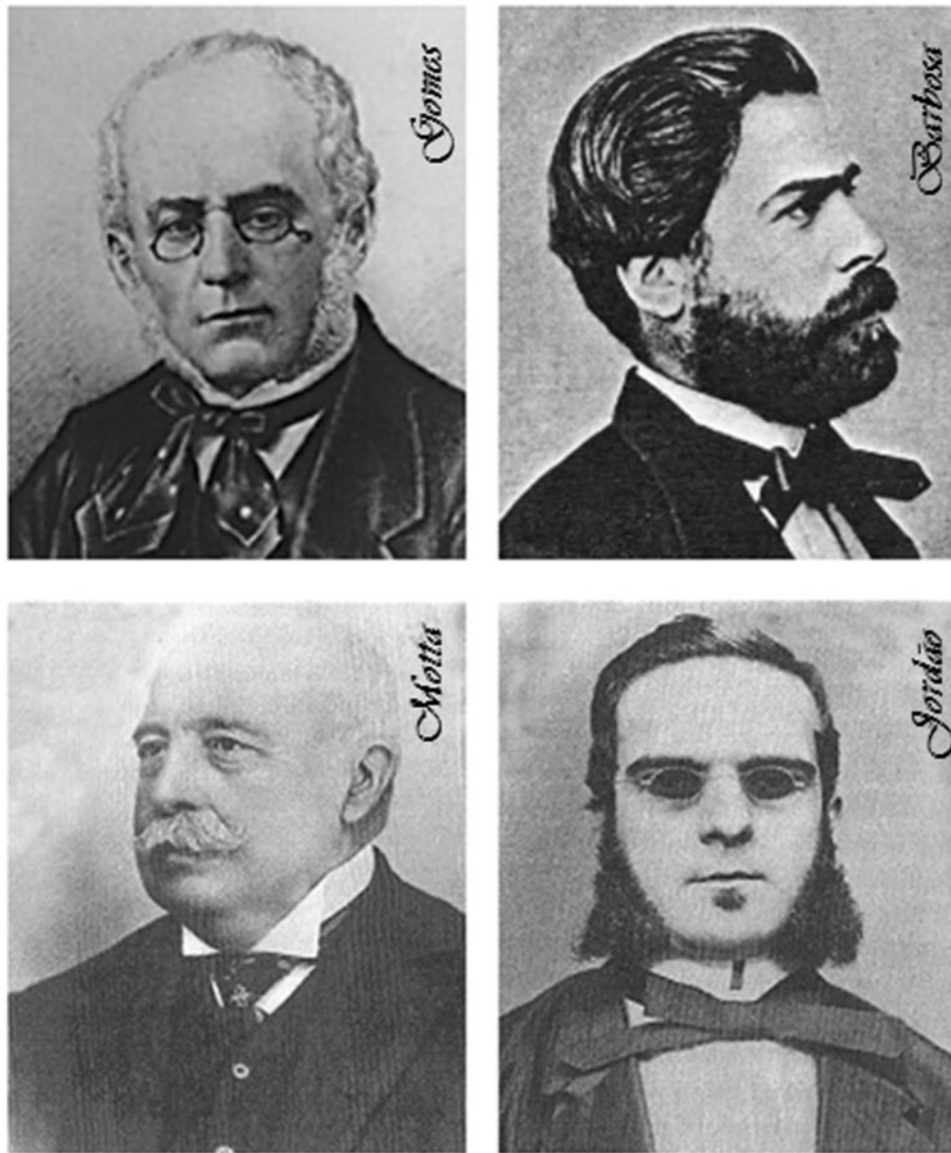
(Gomes, 1865a, b, 1866) were based both on direct observation of the patients in the beginning of the outbreaks as well as on information from the resident asylum physician, Angelo de Sousa. Appearing as his main supporter, Barbosa was one of the rising stars of Portuguese medicine. He was a brilliant surgeon (the first to perform an ovariectomy in Portugal), professor of Pathology and Surgery at the Lisbon Royal Medical and Surgical School and future president of the SCML (1870–72) (British Medical Journal, 1892). His descriptions are based on first-hand observations of the asylum and some of the patients at the end of the epidemic period, for which he provided a detailed clinical picture; he would also outline the theoretical framework for classification of these epidemics of paralysis (Barbosa, 1865, 1866).

The third protagonist in this debate, Abel Jordão, was another of the great clinical figures of his generation and—like Gomes—also trained in Paris. Famous for his *Estudos sobre a Diabete* (Studies on Diabetes, 1864), he would unfortunately die prematurely at age 41 (Lancet, 1874). Also president of the SCML (1862–63), his two publications on this subject (Jordão, 1865, 1866) supply eyewitness accounts of the difficult initial phase of the asylum: 'Soon after the Ajuda asylum was created [...] I started working free of charge in that house [...] As a rule, all children were examined by me before admission, and I had the occasion to note that nearly all [...] were in a terrible state of health. Glandular swellings, more or less pronounced loss of weight and emaciation were almost always present [...]. As far as hygienic conditions the plight of these unfortunate children did not much improve with their admission to this establishment' (Jordão, 1865). He would stop working for the asylum just before the start of the epidemics, in frank disagreement with the poor sanitary and dietary conditions and the excessively religious education the children were subjected to. The final participant in the debate, and the youngest, is Motta, who became a relevant figure of the next generation of physicians [which included Bombarda and Sousa Martins (1843–97)] and who would also be president of the SCML (1879–81). He was the only one of the four who did not have any contact with the patients or the institution, and would intervene twice (Motta, 1865, 1866), mainly to criticize Gomes's interpretation of the facts and to side with Jordão, but did not provide any new data to help judge what happened at the asylum during those 4 years. Therefore, in order to recreate the events during that period, we are limited to the facts that can be derived from the other three authors: Jordão for the background information at the start of the epidemics, Gomes for a description of the initial findings, and Barbosa for a detailed description of the clinical manifestations and asylum conditions at the end of this period. As for the interpretation of the findings, however, all would contribute far beyond their direct experience.

## A succession of epidemics: clinical manifestations

The first outbreak lasted from March 1860 until May 1861 and affected 9 out of the 114 resident female orphans in the





**Figure 2** The protagonists of the debate: Bernardino António Gomes (top left), António Maria Barbosa (top right), Eduardo Motta (bottom left) and Abel Jordão (bottom right).

institution. All were between 10 and 17 years of age and had been at the asylum for more than 2 years. The inaugural complaint was an increasingly severe neuralgic pain in the upper thighs, progressively radiating to the lower limb extremities, accompanied by paresis (patients could not stand or walk, but could move the limbs while lying down) and sensory abnormalities (hypo and anaesthesia) at its peak. In 8 out of 9, both lower limbs were affected, and in the remaining patient both upper and lower right limbs. There were no signs of spinal cord injury such as sphincter abnormalities. A few months later, these findings were joined by 'seizures', 'delirium', emotional lability with excessive crying or laughing and depression. According to the resident physician, contagion by imitation was frequent for these secondary phenomena and they came and went spontaneously, resolving entirely when the orphans were taken out of the asylum. Before

the start of this epidemic, Jordão noted that there was a climate of '[...] sensorial exaltation among the students; they suffered from nightmares, had tingling in the limbs which would eventually become paralyzed, laughed and cried with great facility [and] were impressed by the descriptions of Hell made during religious services [...]. A rumour passed among them that in the basement people were buried and trying to come out. One student, Joaquina Delié, stated at times that she saw people and shadows sitting on her bed, and all her companions started to believe in such phenomena' (Jordão, 1866).

The second epidemic started while the first one was still progressing; in November 1860, 22 internees (16 males and 6 females) between 7 and 15 years of age began complaining of 'crepuscular blindness' or haemeralopia, and xerophthalmia. They were observed by the military ophthalmologist João

Clemente Mendes (1819–75), who went on to describe his findings in his *Estudo sobre a Hemeralopia* [Study on Haemeralopia (Clemente Mendes, 1862)]. All cases resolved spontaneously and without sequelae within a month. Roughly 2 years after the end of the first epidemic the third outbreak occurred: between March 1863 and September 1864, 87 out of the 96 resident orphans developed 'spasmodic' vomiting of poorly digested food, sometimes up to 30 or 40 times per day. Apparently no other clinical manifestations accompanied the vomiting, and all cases resolved when the patients were removed from the asylum and relapsed on their return to the institution.

Finally, in March 1864 the paralysees reappeared temporarily in seven of the patients also suffering from vomiting; five of these had been affected in the first epidemic. The clinical manifestations were identical to the ones in the original outbreak, with the exception that no secondary behavioural phenomena were found. Barbosa provides an extensive description of one of these patients, a 17-year-old girl called Maria Inácia (Barbosa, 1865). He found her in good general condition, with an asymmetric paraparesis (more severe on the right side) and requiring bilateral support for walking '[...] to make a few steps she needs to be held up [and then] will lift slightly each foot, dragging it behind her, keeping it involuntarily turned inwards [...]' (Barbosa, 1865). There were no signs of atrophy in her lower limbs, no reflex reaction to thermal or tactile stimuli, but only to electricity; pain, touch and thermal sensory modalities were deficient from her lower extremities up to her waistline, and she complained of deep, almost constant pain in the gluteal regions. There were no other findings in her neurological or physical exam. When last observed, after leaving the asylum, the patient was reported as being slightly improved, but unfortunately, there is no report of a long-term follow-up.

## The controversy: conflicting pathophysiological concepts

The two central questions debated by the authors were the direct cause and the pathophysiological nature of these epidemics. As to the first, Jordão put great emphasis on the poor hygienic and dietary conditions in the asylum: '[The diet consisted of] a quart of bread at lunch and some tea, some beans and rice at dinner, and some salad and bread at supper [...]' Often I complained about such a dietary system, and managed after a long time to get them to put in 8 pounds of meat in the dinner pot. Because of this state of affairs, I was forced in most occasions to resort to cod liver oil to compensate for nutritional deficiencies and correct lymphatism. [After some time] ulcerative stomatitis rapidly developed and became epidemic [...]' (Jordão, 1865). On the other side, both Gomes and Barbosa reject such accusations, and the latter described in exhaustive detail the daily diet the orphans received e.g. '[...] Bread 416 grams per day. Meat 131 grams five times a week. Codfish 69 grams two times a week. Sugar 30 grams per day [...]' (Barbosa, 1865). He also commented on the building's condition and the state of conservation of the copper cooking equipment and measured the level of plumb in the drinking

water to conclude that in his opinion '[...] Searching for each of the circumstances that most directly influence the life and health of individuals, air, water, food [...]' nothing stands out which is not found to a much larger extent in the dispersed poor population of Lisbon [...]' (Barbosa, 1865). These two apparently conflicting visions probably just reflect differences in the time of observation; Jordão's description is most relevant for the earlier years of the institution and therefore bears a more direct causal relationship with the start of the epidemics.

Beyond dietary problems, Gomes also pointed to several 'moral causes', such as the life of confinement, overcrowding and the effects of religious education. As we saw, Jordão was extremely critical of what he called the 'excessive religious pressure' to which the orphans were subjected. Barbosa was also in agreement with the negative effects of overcrowding and co-habitation on several young and impressionable young patients. In their joint opinion, these were sufficient to induce a 'state of susceptibility to nervous disease' which Gomes and Jordão called 'hysterism' while Barbosa preferred the term 'nervosism'. At that time, as we will see, this distinction was more than just semantic.

The second set of questions relate to the nosological classification of the paralysees. Barbosa provided the best systematization and an overview of the contemporary conceptual framework. Paralysees could have organic causes e.g. direct spinal cord lesions, ('material lesions [...] which attack the spinal cord primarily or secondarily, either in its sheathing or the spinal tissue itself'), ischaemic causes ('lack of irrigation of the spinal cord and nerves [...] the organic condition for ischaemic paraplegia is the obstruction in the abdominal aorta artery which interrupts the access of blood to the lower spinal cord and lower limb nerves'), dyscrasic causes ('alterations in the qualities of the blood', such as 'chlorosis', intoxications with mineral—'lead, mercury, arsenic'—and vegetal—'poison mushrooms, tobacco, camphor, ergot, lathyrus sativus'—substances) and functional or 'nervous' causes ('paralysees due to a functional perversion, that is, in which there is no appreciable material lesion of the nervous centres, or a qualitative or quantitative alteration in the blood'). This last category did not refer, as today, to purely psychosomatic diseases, but brought together paralysees caused by pyrexia and acute diseases, cachexia, neurosis and hysteria, and the recently described reflex and peripheral paralysees caused by 'prolonged excitation transmitted to the spinal cord by the peripheral nerves of the excited organs such as the genito-urinary tract, abdominal organs, the skin, etc.' (Barbosa, 1865).

All authors agree that the events in the Ajuda asylum should be classified within the functional category. The diagnosis of chlorosis, or chloro-anaemia, was also briefly considered; this disease, described for the first time in 1615 by Jean Colliot de Varanda (c. 1563–1617) was later identified in part with hypochromic anaemia. It was thought to attack predominantly young adolescent virgin women of 'weak constitution' (Mercer and Wangenstein, 1985), but for Thomas Sydenham (1624–89) and Armand Trousseau (1801–67) chlorosis was a nervous disease that supposedly caused a variety of symptoms, such as alterations in skin tone (giving it a green tinge, hence the name, *khloros*—*χλωρος*—meaning 'greenish-yellow'), lack of energy, dyspnoea, dyspepsia, headaches and amenorrhoea, and might cause paralysis by altering

the proportions of blood constituents (dyscrasia, a term adapted from the hippocratic-galaenic concept of unbalanced humours). As regards the Ajuda epidemics, Barbosa clearly opposed the diagnosis, based on the facts that 'the general status of the paraplegics, their physiognomy was certainly not that of anaemic or chlorotic patients [...] they were not pale, or discoloured, or had the bad colour of chloro-anaemic girls [...] they never had heart palpitations, disturbances in digestive function, or oedema. [...] The ones who had menses continued to have them without any alteration. [...] Some presented with a carotid murmur, [but] that sign, in isolation [...], does not appear to us to indicate the presence of this disease' (Barbosa, 1865). But even though there was agreement in classifying the paralyses within the functional category, there was dissent as to the precise diagnosis. For Gomes they were reflex paralyses, Barbosa preferred to call them peripheral, and for Jordão and Motta they were hysterical. Again what *a priori* seems to be a byzantine debate over small differences in meaning actually reflects different pathophysiological concepts, which became the fulcrum of the debate: hystericism versus nervosism, reflex and peripheral versus hysterical paralyses.

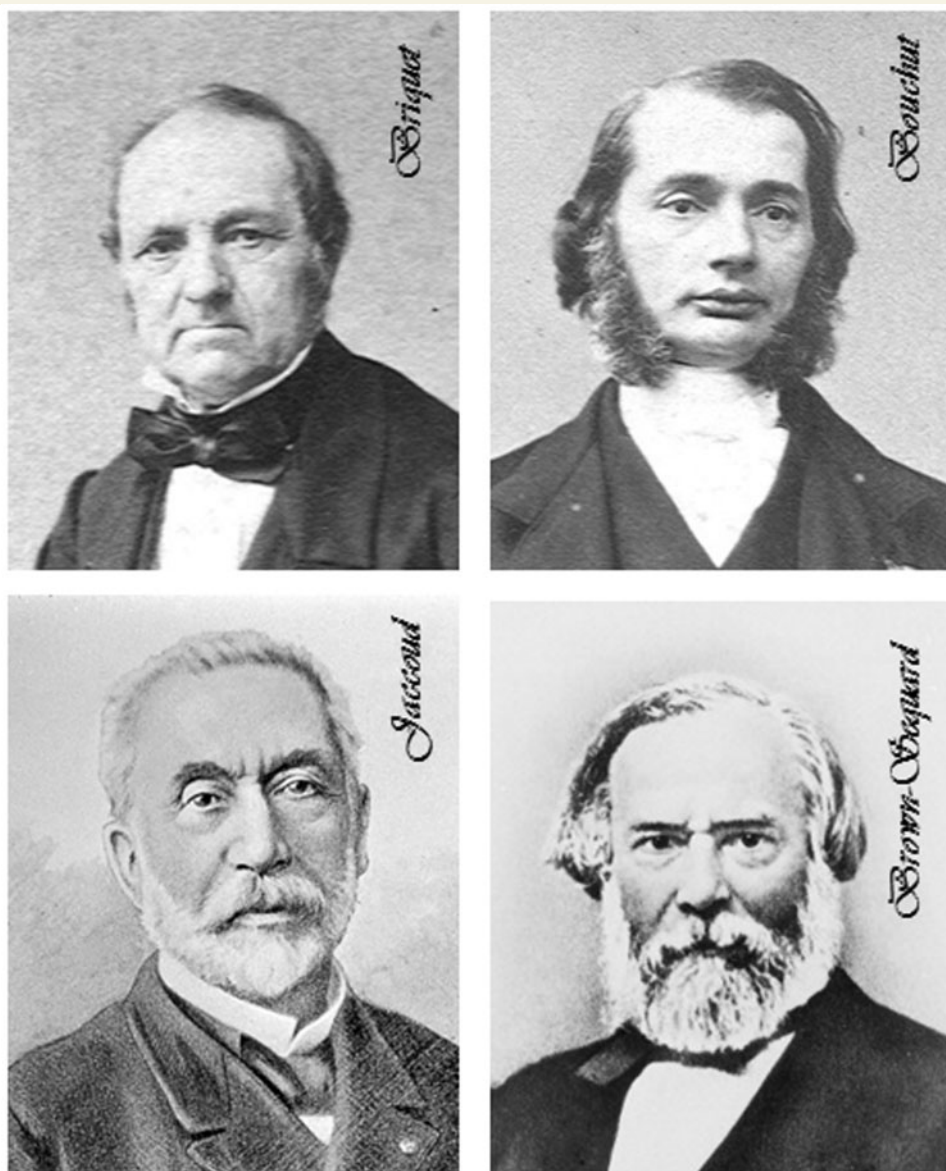
The concept of nervous diseases, or 'neurosis', first appeared in the late-17th century in William Cullen's classification as a separate class of general diseases to encompass 'all those preternatural affections of sense and motion, which [depend] on a more general affection of the nervous system' [quoted by (Lopez Pinero, 1983a)]. By the mid-19th century, neurosis had evolved, via the works of Philippe Pinel (1745–1826), Étienne Jean Georget (1795–1828) and Achille Louis Foville (1799–1878), to have both an anatomoclinical as well as a functional meaning. In this view, neurosis came to signify a group of diseases of unknown cause, whose symptoms pointed to an origin in the nervous system, but for which no identifiable pathological basis could be found (Lopez Pinero, 1983b). While still grouping together several entities, which by modern standards would not be included in a list of nervous system disorders (such as Foville's Order IV 'neuroses of nutrition', which included disorders of digestion, breathing and circulation, or Georget's inclusion of asthma, nervous palpitations and gastralgia), the list became progressively narrower and reflected functional localization to the nervous system.

Hysteria and hystericism paralleled the evolution of the concept of neurosis. Deeply rooted in classical medical tradition, the diagnosis of hysteria had been overly used as an explanation for mood and behaviour abnormalities associated with a diverse array of organic dysfunctions, typically in young females with menstrual or sexual problems, and thought to be directly linked to problems in the female sexual organs. This view was to change gradually into one of a disease that could affect both genders and appear before puberty, without an exclusive relationship to sexual or gynaecological problems, and caused by a poorly defined nervous dysfunction. In fact, as far back as the 17th century, both Sydenham and Thomas Willis (1622–75) had started to break with the traditional view and proposed that hysteria was due to dysfunction of the nervous system. Hysteria subsequently appeared within Class III (*Spasmi*) in Cullen's classification of the neuroses, Robert Whytt (1714–66) named it as one of the 'simple nervous disorders' (Lopez Pinero, 1983a) and in Pinel's

classification it is listed again in the class of neuroses, in Order V ('neuroses of sexual function') (Lopez Pinero, 1983b).

Even if placed within the group of nervous diseases, the conceptual latitude of the diagnosis of neurosis in the early 19th century still allowed two competing pathophysiological interpretations of hysteria, in which the ultimate cause of the disease was either still in the sexual organs indirectly linked by peripheral nerves to the nervous system ('uterine neurosis'), or primarily in the brain ('encephalopathic') as was defended by Georget (Lopez Pinero, 1983b). Later, Pierre Briquet (1796–1881) (Fig. 3) attempted to steer away from these localizationist discussions by studying hysteria with the great methodical spirit of 19th-century French physicians, and managed to collect and systematize the full clinical histories of 430 patients with the diagnosis of hysteria (Goetz *et al.*, 1995). In Briquet's view, hysteria was also a 'cerebral neurosis' in which affective problems manifested themselves indirectly as varied dysfunctions of multiple organ systems (Mai and Merskey, 1981), but he negated the supposed protean and unpredictable symptomatology of the disease (Lopez Pinero, 1983b). In his *Traité de l'Hystérie* (Briquet, 1859), he methodically described the spectrum of clinical manifestations of hysteria (hyperaesthesia, anaesthesia, sensory perversions, spasms, convulsions, hysterical paralyses, disturbances of muscle contraction), predisposing factors (psychological and physical, social class, education, emotional and moral problems, profession, sexual habits), precipitating factors, clinical course, diagnosis and prognosis (Mai and Merskey, 1980). When, a decade later, Sigismond Jaccoud (1830–1913) (Fig. 3) published his *Traité de Pathologie Interne* (1869), the neuroses were classified in a purely localizationist framework as cerebral, cerebrospinal, brainstem or peripheral, and hysteria placed in the cerebrospinal category (Jaccoud, 1869). Charcot would further develop this concept of hysteria as an organic brain disease due to a 'functional' disturbance of the cortex (functional since, as with epilepsy, he could not find any microscopic abnormalities therein) (Goetz *et al.*, 1995). For him, there was no real difference between the clinical characteristics of sensory and motor symptoms (such as paralysis) caused by hysteria or structural lesions, as both were related to dysfunction of the same pathways. It was only with his disciple, Joseph Babinski (1857–1932), that the distinction between functional and organic neurological symptoms became clearer. Babinski (and his contemporaries) helped detail neurological semiology, including the description of the cutaneous-plantar response (and its association with pyramidal tract lesions), the cremasterian and abdominal-cutaneous reflexes, and the precise topography of hysterical hemiplegia and hemianaesthesia (Philippon and Poirier, 2008). These differences suggested a psychological causality for hysterical symptoms and would finally lead Babinski to propose that hysteria was a psychical state in which the patient had a pathological predisposition to self-suggestion, and that its name should be changed to pythiatism (Babinski and Froment, 1917). So, at the time of the SCML debate, hysteria was being increasingly considered as a functional but organic brain disorder that might be precipitated by emotional problems, religious pressures or malnutrition; Gomes, Barbosa and Jordão quote Briquet and evidently use the term in that context.





**Figure 3** The influences on the debate: Pierre Briquet (top left), Eugène Bouchut (top right), Sigismond Jaccoud (bottom left) and Charles-Édouard Brown Sequard (bottom right).

In comparison, the competing concept of nervosism (or 'nervousness'), supported mainly by Barbosa, was a relatively new and fashionable designation proposed in 1860 by Eugène Bouchut (1818–91) (Bouchut, 1860) (Fig. 3). He defined this condition as a 'general neurosis' (which meant not specifically located in the central nervous system) characterized by an association of several functional disturbances of sensibility, cognition, movement and the main organ systems (Winslow, 1860). Nervosism could be classified as cerebral, spinal, laryngeal, gastric, uterine, cutaneous, spasmodic, paralytic or painful, depending on its manifestations, although underlying all subtypes was a state of chronic depletion of a supposed 'nervous force'. As we can see, this is a much more vague concept in comparison with the clinical rigour of Briquet's hysteria and countercurrent to the contemporary movement towards functional localization of neuroses, and was not widely

accepted even by his contemporaries (Winslow, 1860). Barbosa uses the concept mainly as supporting evidence for the existence of a 'nervous exhaustion' which could cause the peripheral type of paralysis. By the end of the 19th century, nervosism was lumped together with other so-called 'intermediate neuroses' (such as spinal irritation) and with George Miller Beard's (1839–83) neurasthenia, and together with hysteria these were the two major neuroses that survived into the next century. They became the focus of debate between the schools of the Salpêtrière [Charcot and his disciples such as Babinski and Pierre Janet (1859–1947)] and Nancy [represented by Ambroise-Auguste Liébeault (1823–1904) or Hypollite Bernheim (1840–1919)], culminating in the 'psychological period' of interpretation of which Sigmund Freud (1856–1939) is the most evident representative (Lopez Pinero, 1983c).

As far as the nosological classification of the paralyses, the pathophysiological distinctions between the above mentioned 'functional' paralyses are intriguing. The existence of hysterical paralyses was a well-documented phenomenon among other motor and sensory manifestations of hysteria and could have as direct causes hysterical seizures, sudden 'moral perturbations', excessive fatigue, abrupt menstrual suppression or 'excessive evacuations' (Lebreton, 1868). Abel Jordão had no doubts that all the epidemic outbreaks observed were '[the] progeny of hysteria [...]'. (Jordão, 1865) and Motta fully supported this idea. Both base their conclusions on the social circumstances surrounding the admission of the children to the asylum, the dietary and hygienic conditions, rigorous education and excessive religious practice, which would contribute to the creation of a 'hysterical constitution'. Even the initial clinical complaint—pain—was judged to be a 'hysterical arthralgia' or 'myosalgia'. Admittedly, there were other phenomena, such as the ocular findings, which could not be explained so simply.

The second entity under consideration was the so-called 'reflex paralyses' that had been recently proposed by Charles-Édouard Brown-Séquard (1817–94) (Brown-Séquard, 1861) (Fig. 3). In reflex paralysis, a peripheral lesion causing excessive irritation of sensory nerves induced a reflex vasoconstriction of spinal cord pial vessels at the level of the injury, resulting in a temporary loss of adequate blood supply that would therefore cause motor paralysis and sensory abnormalities (Lopez Pinero, 1983c). Years before, Foville had already proposed such a causal link between nervous dysfunction and the status of blood flow in the nervous system (Lopez Pinero, 1983b). Brown-Séquard went further, producing original experimental animal data in which stimulation of peripheral nerves caused spinal vasoconstriction, and citing several supporting clinical phenomena such as paraplegia after renal inflammation, lesions of the uterus or intestine, hemiplegia caused by pneumonia, blindness resulting from frontal nerve lesions, limb paralysis after gunshot wounds and so-called 'reflex inflammations' (Brown-Séquard, 1861). In the Ajuda epidemics, the inducing phenomenon would be the gluteal pain which '[...] by an anomalous neuralgia of the posterior branch of the last lumbar roots [...]'. (Gomes, 1865b) would result in a dysfunction of the lumbar spinal cord. Haemeralopia might be a reflex paralysis of vision caused by excitation of the frontal nerves by the '[...] intense gas lighting in the Asylum [...]'. (Barbosa, 1865), and the vomiting a similar phenomenon caused by irritation of gastric nerves.

Gomes was the main defender of this theory and Motta its main opponent. In fact, he goes as far as emphatically saying that 'This new nosological entity seems to me an exalted concept of the author, a truly utopian idea [...] I think this doctrine unsustainable before reasoning, and in the presence of physiology [...]'. (Motta, 1865), and more diplomatically: 'It seems to us that this doctrine can never be grouped with the most beautiful conquests of the human spirit, which before being received and acclaimed had to fight fiercely to vanquish violent opposition. [...] If [Brown-Séquard] was not for so many reasons a respectable and known doctor, he would surely not build his reputation by inventing this reflex paralysis fable [...]'. (Motta, 1866).

Finally, and although taking a similar view to Gomes's that hysteria was a concept too vague and abused to be useful, Barbosa preferred to classify the clinical phenomena under the designation of peripheral paralyses that Jaccoud had recently proposed (Jaccoud, 1864). Unlike the modern concept of peripheral paralysis, Jaccoud was not referring to lesions of the peripheral nerves or nerve roots; instead, according to him, such paralyses occurred when excessive irritation of the peripheral nerves led to an exhaustion of the capacity of the spinal cord to transmit nerve impulses, 'un épuisement de l'excitabilité des centres nerveux par une excitation continue' or 'neurolysis' (Dechambre, 1885). As we saw above, Jaccoud would go on to propose the existence of a category of 'peripheral neuroses' which included cases of neuralgia, anaesthesia, hyperkinesia and akinesia (Lopez Pinero, 1983b). Barbosa's theory, therefore, is that nervosism had led to a depletion of nervous 'energy', which, compounded with excessive peripheral excitation, caused a full-blown central nervous system dysfunction resulting in varied clinical manifestations of which paralysis was the most extreme.

## The Ajuda epidemics: modern view and alternative explanations

It must be emphasized that this debate is very much a product of its historical moment. The diversity of clinical entities and proposed diagnosis seem confusing to the modern reader, unclear and imprecise, incompatible with the pathophysiological framework that we nowadays recognize. They do, however, provide a portrait of mid-19th-century neuropsychiatry, a period that was witnessing major conceptual changes in this clinical area. It should be said that the contemporary view of these types of epidemics was also very much influenced by two other similar outbreaks in preceding decades in France, which were frequently referred to during the debate. First, the so-called 'Paris epidemic' of acrodynia in 1828–32, which consisted of pain and paresthesias in the extremities (mainly lower limbs), associated with other manifestations such as paralysis and cutaneous lesions in the same locations and gastrointestinal problems (Genest, 1829); and second, the epidemic of 'catalepsy' and sensory problems occurring in 1847 in the *Maison de Refuge du Bon Pasteur* of Amiens, in which 22 females were affected (Sandras, 1851). As in the Ajuda epidemics, the circumstances appeared similar: an outbreak of 'nervous' disease, combining motor and sensory problems, having in common its appearance in a religious community; similar explanations were provided for both these episodes, and they were finally attributed to hysteria and labelled as 'nervous epidemics'. This was seen at the time as an argument for favouring a similar interpretation for the Ajuda epidemics.

One interesting realization is that in no case do the authors refer to the potential role of direct lesions in the peripheral nervous system in discussing these epidemics. Even Jaccoud's theory of peripheral paralysis is entirely alien to our present view of peripheral nerve disorders and a direct heir of Broussais's concept of 'morbid sympathies' (Lopez Pinero, 1983b, c). Simply, peripheral



neuropathy was a nosological entity under development exactly during this period, and the debate over the Ajuda paralyse coincides in time with the recognition of the new category of neurological diseases of the peripheral nerves. The existence of diseases affecting several peripheral nerves simultaneously, or 'multiple neuritis', would only be proposed by Ernst von Leyden (1832–1910) in 1874 (von Leyden, 1874). Previously, in 1843, Robert Graves (1797–1853) had already anticipated that lesions in peripheral nerves could cause paralysis and sensory dysfunction of the limbs in his comments on the Paris acrodynia epidemic: 'The French pathologists, you may be sure, searched anxiously in the nervous centres for the cause of this strange disorder, and could find none; there was no evident lesion, functional or organic, discoverable in the brain, cerebellum or spinal marrow [...] Can anyone [...] hesitate to believe that paralysis [...] may arise from disease commencing and originating in the nervous extremities alone?' (quoted from Pearce, 1990). Also, about the same time, in 1859, Jean Baptiste Octave Landry de Thézillat (1826–65) had published the first volume of his *Traité complet des paralysies* and his work on ascending paralysis, and 5 years later Louis Duménil (1823–90) would confirm the presence of lesions of the peripheral nerves in such patients (Brody *et al.*, 1994; Pearce, 1997). It is curious that although the Portuguese physicians knew of Graves's work (quoted, for example, by Gomes and Barbosa), none mentioned the possibility of a peripheral nerve lesion in the Ajuda cases.

Is it possible today to propose an alternative interpretation for the events in the Ajuda asylum between 1860 and 1864? Assuredly, given the uncertainties regarding the true clinical characteristics of the outbreaks, the easiest explanation may be simply to invoke the all-encompassing label of somatization disorder, epidemic hysteria or mass sociogenic illness. But although single episodes of motor paralysis of psychosomatic (functional) cause are relatively common in neurological practice, what is the evidence regarding multiple simultaneous cases? In a thorough historical review of such events, Sirois collected and systematized the main characteristics of 78 epidemic hysteria outbreaks between 1872 and 1972 (including a reference to Jordão's publication) (Sirois, 1974). In his classification, the Ajuda paralyse would belong to the category of cumulative outbreaks in which clinical manifestations typically involve less than 10 persons, in institutional environments and develop over 2 weeks to 1 month. Overall, the largest number of cases of epidemic hysteria in this series occurred in schools ( $n=34$ ), involved between 10 and 20 people ( $n=25$ ), below 20 years of age ( $n=41$ ), in females ( $n=58$ ) and lasted from 3 to 14 days ( $n=28$ ). That seems to partially fit with the profile of the Ajuda epidemics, with the exception that long duration (over 1 month) outbreaks are more rare ( $n=15$ ). However, symptoms clearly differ: seizures ( $n=19$ ), abnormal movements ( $n=14$ ), fainting ( $n=9$ ) and paresthesia and anaesthesia ( $n=9$ ) were the most common, whereas outbreaks of paralysis were much less frequent (only three cases, ~4% of the total). In a more recent work, in which 70 outbreaks from 1973 to 1993 were studied, there are no major changes in their general characteristics, but the clinical manifestations change slightly; the percentage of paralysis cases increases to 11% ( $n=7$ ) and there is a complete disappearance of seizures and abnormal movements

(Boss, 1997). It is well known that social and cultural influences throughout time have a significant impact on clinical manifestations of mass sociogenic illness (Bartholomew and Wessely, 2002). We can conclude from these reviews that epidemics of hysteric paralysis are not common now and were even less so in the 19th century, leaving the field open to alternative explanations.

So far we have focused mainly on the paralysis outbreaks, which may or may not have been caused by hysterical conversion. However, it is much less probable that the epidemic of vomiting, which affected nearly the totality of the subjects (90% of the resident population), might also be due to the same cause. Even in hyperemesis gravidarum, a well-recognized phenomenon in pregnant women and one of the most common causes of recurrent vomiting, support for a psychosomatic causation has decreased markedly in recent years (Goodwin, 2008). Furthermore, it is hard to accept that 'hysterical' vomiting, even if present in non-pregnant young females and males, could assume epidemic proportions and be maintained throughout such a prolonged period of time. Epidemic vomiting is usually related to gastrointestinal infections (e.g. winter vomiting disease caused by Norwalk virus infection) or intoxications. With that in mind, an interesting piece of evidence, briefly mentioned by Gomes, is that the vomiting epidemic was transmitted to another institution (the Junqueira asylum) when some of the affected children were transferred there (Gomes, 1865a). This clearly points to an infectious cause, which also fits in with the poor hygienic conditions and overcrowding in the Ajuda asylum.

Finally, there remains to explain the epidemic of night blindness, or haemeralopia. As we saw, the clinical characteristics of these patients were best described by João Clemente Mendes, who highlighted the presence of both haemeralopia and xerophthalmia, and compared his findings to what Charles Deval (1806–62), in his 1844 *Traité de Chirurgie Oculaire*, had called xeroma. The concurrent observation of night blindness and conjunctival dryness would be repeated 3 years later by Pierre Bitot (1822–88), who left his name connected with the patches of keratinized metaplasia in the bulbar conjunctiva that are nowadays considered pathognomonic of vitamin A deficiency. In fact, Mendes's work has been referred to as one of the first descriptions of avitaminosis A, together with those by Mecklenburg in 1855 and Anton von Huebner (1822–73) in 1860 (Semba, 2007). Taking into consideration the dietary deficiencies pointed out by Jordão, it is not strange that such cases could have appeared in the Ajuda asylum.

In fact, nutritional deficits could also have been at the root of the outbreaks of paralysis, since we now know that vitamin deficiencies are frequent causes of peripheral nerve damage. Historically, the first description of a polyneuropathy was made (posthumously) in 1642 by Jacob de Bondt [Bontius (1592–1631)] in beri-beri patients (Pearce, 1990), and the Paris acrodynia epidemic was compared from the start to the manifestations of pellagra, as Gomes also points out (Gomes, 1865a; Wood, 1921). Furthermore, at the end of the 19th-century Henry Strachan described an epidemic of sensory and optic neuropathy, ataxia, hearing problems, cortico-spinal dysfunction and stomatitis in Jamaican sugar cane workers (afterwards named

Strachan's syndrome), probably caused by chronic malnutrition (Strachan, 1897). Similar observations were made by Domingo Madan (1856–98) in Cuba during the rebellion against Spanish domination between 1886 and 1898 (Santesteban-Freixas *et al.*, 1997). Recently, another such epidemic struck Cuba, manifesting itself as a sensory and optic neuropathy, neuro-sensorial deafness, dorso-lateral myelopathy, spastic paraparesis, dysphonia and dys-autonomy (Thomas *et al.*, 1995). Therefore, the events at the Ajuda asylum could be one of the earliest modern descriptions of avitaminosis-related peripheral nerve disease (probably some form of sensory-motor radiculoneuropathy), preceding those of Strachan and Madan by a few decades. Alternatively, the presence of behavioural abnormalities in conjunction with the remaining clinical findings should also raise the possibility of a pellagra outbreak similar to the Paris epidemic. In fact, pellagra was endemic in Portugal at the time of the debate and its neurological manifestations were described by Bombarda before the end of the 19th century (Bombarda, 1896). In either case, the contributions of Gomes, Barbosa and Jordão to the field of nutrition-deficient neurological diseases deserve to be remembered.

In light of these interpretations, the picture that emerges for the period of time between 1860 and 1864 is not one of successive outbreaks of epidemic hysteria, but rather that of a malnourished and overcrowded community of children and adolescents living in poor hygienic conditions, and who suffered from diseases typically associated with these circumstances. There is some supportive evidence for this hypothesis. It is obvious that the affected orphans went through a prolonged period of economic deprivation: after the death of their parents in 1856–57 and during the initial turbulent years at the asylum (as documented by Jordão) they may have been subjected to several years of malnutrition. When Barbosa, in 1865, carefully documented their dietary intake (which by then appeared adequate), almost a decade had passed since the yellow fever epidemic. This period of time would be sufficient to induce the clinical manifestations we now know to be related to vitamin deficiency, such as painful peripheral neuropathies and haemeralopia. At least three pieces of clinical evidence are concurrent with this interpretation: first, Jordão's eyewitness description of the clinical status of the orphans at the time of admission into the asylum: 'lymphatism', 'pronounced loss of weight and emaciation' and 'ulcerative stomatitis rapidly developed and became epidemic', as well as his record of the truly deficient dietary regimen, which besides being hypocaloric was notably absent in several food categories, including fresh fruits and vegetables containing vitamins and trace elements; second, Clemente Mendes's pioneering description of the ocular and visual findings now known to be associated with vitamin A deficiency during the second epidemic; and third, despite the gradual improvement in the dietary conditions at the asylum that could have helped reverse the clinical situation, the reappearance of paralysis cases in patients suffering from chronic vomiting, again pointing towards the role of malnutrition. In fact, chronic vomiting has been associated with the acute appearance of neurological symptoms such as Wernicke encephalopathy (Wilson *et al.*, 2006). Naturally, it is still possible that some, if

not all, of the clinical events during those 4 years might have been psychosomatic in nature, or at least formed by a superimposition of hysterical colouring over an organic substrate, but given the circumstantial evidence, it is more reasonable to blame them entirely on the deleterious consequences of poor social support: poverty, malnutrition and institutional overcrowding.

## Conclusions

There are two main lessons to be taken from the 'Ajuda paralyses' debate. On one side, they provide insight into the status of familiarity and understanding of diseases of the nervous system by Portuguese clinicians in the mid-19th century. At this time, Rilhafes was the single institution in the country for the treatment of neuropsychiatric patients and there were no dedicated clinics or academic researchers in these areas. It is therefore notable that Gomes, Barbosa, Jordão and Motta were so well aware of the latest scientific developments and had their own critical views on them. The debate in the SCML reveals the interest that these nascent disciplines had for Portuguese clinicians. Barbosa, Jordão and Motta, as respected teachers at the Lisbon Medical School, would help foster the development of the next generation of clinicians, such as Bombarda and Sousa Martins, towards the formal establishment of neurology and psychiatry as independent disciplines in Portugal.

On the other side, this debate illustrates the development of the modern concepts of psychosomatic disease and peripheral neuropathy, from the overabundance of other confounding nosological entities that did not survive the test of time, such as Bouchut's nervosism, Brown-Séquard's reflex paralyses and Jaccoud's peripheral paralyses. At a time when the clinical-anatomic method and the development of neuropathology were finally providing explanations for the mysterious manifestations of neurological and psychiatric diseases, these vague concepts and hypothetical pathophysiological constructs were naturally condemned to non-existence. Biological understanding of the mechanisms of neurological disease would become the core feature which allowed the development of effective therapies, a process that accelerated in the last decades of the 20th century. Psychiatry, fortunately, is currently undergoing the same type of scientific revolution, based on the recent availability of tools—genetics, experimental pharmacology and functional imaging, to name a few—that allow us to pry into the processes underlying functional brain disorders. Hopefully, this will help end the century-long artificial division of neuropsychiatry into two separate fields, bringing us full circle to the 19th-century holistic view of 'nervous diseases', only now based on a solid foundation of neuroscientific knowledge.

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